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BEFORE THE BOARD OF ~~APPEALS AND~~ INTERFERENCES
IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

In re application of: Preet Chaudhary

Serial No. 09/490,187

Filed: January 23, 2000

For: *Gene Expression in Ectodermal Dysplasia*

Group Art Unit: 1635

Examiner: McGarry, Sean

Attorney Docket No.UTSD:0680

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Signed _____
Richard Osman

REPLY BRIEF ON APPEAL

The Honorable Board of Appeals and Interferences
United States Patent and Trademark Office
Washington, D.C. 20231

Dear Honorable Board:

The Examiner's Answer mailed November 19, 2001 does not accurately represent the record and does not address the subject claims.

Claims 1-8 require detecting the presence of or predisposition to an ectodermal disorder by (a) detecting the presence of a human TAJ gene or gene product in a cell; and (b) correlating the presence of the TAJ gene or gene product with a presence of or predisposition to an ectodermal disorder. The Specification thoroughly teaches and exemplifies the method defined by these steps, readily enabling one of ordinary skill in the art to practice the method as claimed without undue experimentation. For step (a), the Specification describes a variety of suitable detection methodologies (p.4, lines 9-28; p.6, lines 3-13), teaches a large panel of exemplary TAJ specific probes (allele-specific antibodies and hybridization probes; p.4, line 31 - p.6, line 2), and provides detailed exemplification of detection by in situ and chromosomal hybridization (p.9, lines 3-17), TAJ allele-specific PCR amplification (p.12, lines

5-22), transcriptional reporter assay (p.10, lines 6-29), and immunocytochemistry (p.14, line 29 - p.15, line 5); see also p.17, lines 14-31. Step (b) involves no more than correlating the detected TAJ gene or gene product with an ectodermal disorder. In many cases, this entails no more than cross-referencing to a known clinical correlate. The Specification describes alternative means to implement this step (p.6, lines 14-26), teaches a large panel of TAJ genes and gene products associated with an ectodermal disorder (p.3, line 16 - p.4, line 3) and provides detailed exemplification of correlation by chromosomal mapping (p.9, lines 12-17), animal model (p.9, line 18 - p.10, line 3) and clinical diagnosis (p.12, lines 5-22).

Both required steps of these TAJ detecting claims are thoroughly taught, described and exemplified, fully enabling one skilled in the art to practice the claimed invention without undue experimentation. The Answer's criticisms of the TAJ detection data reported in the Specification, and particularly the reporting of qualitative as opposed to quantitative data, are believed to reside outside the bounds of a proper enablement analysis duly limited to the recited claims. For example, Table 2 provides exemplary allele-specific TAJ antibodies and allele-specific hybridization probes. Rather than reciting quantitative data that cannot be compared across experiments, the Table indicates demonstrable allele-specific antibody binding with a normalizing "+++" designation, and demonstrable allele-specific hybridization with a "+++" designation. The qualitative "+++" indicates that unequivocal allele-specific binding or allele-specific hybridization is obtained. Specific-binding/hybridization are readily assayed by those skilled in the art, and exemplified in our Specification, e.g. Examples III and IV. Similarly, we believe the Answer's statements regarding the correlation step presume that the claims require more than the recited step. Note that the entire detection method is exemplified in Example IV, wherein the practitioner detects the presence of the human TAJ gene product (by PCR) and correlates that presence with an ectodermal disorder (Clouston syndrome).

The Answer's suggestion that correlating ectodermal disorders that may be associated with TAJ mutations would involve undue trial and error appears to read too much into the required claim step. Our claims do not require correlating every ectodermal dysplasia with every TAJ mutation, but merely detecting *a* TAJ mutant and then correlating *that* mutant to the presence of *an* ectodermal disorder. Whether the number and potential causes of ectodermal disorders are 5 or 500 is submitted to be not relevant to our claims. Note, for example, the exemplification shown in Example IV (p.12,

lines 5-22). There is no trial and error – the cell comes from a particular source (e.g. patient) having a particular clinical presentation, to which the practitioner is not blind. In fact, a particular clinical presentation of ectodermal disease is the reason the patient's TAJ gene is being analyzed. In *In re Wands*, the enablement issue was not whether it would require undue experimentation to make all the possible antibodies having the required affinity, but rather whether it would require undue experimentation to make a given such antibody. Similarly, here the issue is not whether it would require undue experimentation to analyze the correlation of every possible TAJ mutant with every possible ectodermal disorder, but rather whether it would require undue experimentation to analyze the correlation of *a* TAJ mutant to *an* ectodermal disorder - and one single disorder will suffice for our claims.

Though there is no evidence to the contrary, we have provided an expert Declaration under 37CFR1.132 documenting that the Specification readily enables one of ordinary skill in the art to practice this two-step detection method as claimed without undue experimentation. Hence, the *uncontroverted* evidence of record demonstrates compliance with 35USC112, first paragraph.

In his Answer, the Examiner repeatedly mistates our claims, flailing at strawmen of his own creation.. We are not claiming "any and all ectodermal dysplasias"; we do not require determination of "over 150 know [sic][1] ectodermal disorders". Again, the claims 1-8 require only two steps: a) detecting the presence of a human TAJ gene or gene product in a cell; and (b) correlating the presence of the TAJ gene or gene product with a presence of or predisposition to an ectodermal disorder. To practice the method, the practitioner first detects the TAJ gene or gene product in a cell of the patient. If present, the practioner then determines whether patient has an ectodermal disorder (or is predisposed to one). If so, the practitioner merely notes the correlation - that of the TAJ expression with the presented clinical manifestation. That is it. The practitioner does not require any external information about hundreds of ectodermal dysplasias and is not required to do any research at all correlating TAJ expression and such dysplasia. The only correlate required is that inherently presented with the subject patient.

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[1] The Answer's repetitive use of this misspelled phrase (p.6, line 8; p.6, line 14; p.7, line 4; p.7, line 7) worriedly suggests he is just copying and pasting the same argument over and over again.

The Answer's frightening depiction of endless research[2] is indeed intimidating, but it has no relevance to our claims.

Claims 9-21 require modulating the functional expression of a TAJ gene or gene product in a cell by contacting the cell with an agent which specifically binds and modulates the functional expression of a human TAJ gene or gene product, wherein (a) the cell is an ectodermal cell; or (b) the cell is a germ cell which gives rise to progeny ectodermal cells and further detecting the functional expression of the TAJ gene or gene product in the progeny cells.

The Specification thoroughly teaches and exemplifies the method defined by these steps, readily enabling one of ordinary skill in the art to practice the method as claimed without undue experimentation. The Specification explains how this method is implemented, including its application to germ cells which give rise to progeny ectodermal cells (p.6, line 29 - p.7, line 9), describes a variety of suitable TAJ binding and modulatory agents (p.7, lines 10-19), teaches a panel of exemplary agents shown to allele-specifically modulate functional expression of a TAJ gene or gene product (p.7, line 21 - p.8, line 18), describes how these agents are delivered to the cell (p.8, lines 20-30), and provides detailed exemplification of the method as applied to human keratinocytes in vitro and in vivo (Examples V and VI, p.12, line 24 - p.17, line 31).

The required step(s) of the TAJ modulating claims 9-21 are thoroughly taught, described and exemplified, fully enabling one skilled in the art to practice the claimed invention without undue experimentation. The Answer's criticisms of the data reported in the application, and particularly the reporting of qualitative as opposed to quantitative data, are believed to reside outside the bounds of a proper enablement analysis duly limited to the recited claims. For example, Table 3 provides exemplary agents which allele-specifically modulate TAJ expression. Rather than reciting quantitative data that cannot be compared across experiments, the Table indicates demonstrable allele-specific expression

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[2] "One skilled in the art is left to determine temporal, developmental, quantitative or qualitative TAJ misexpression and the wide variety of causalities that may effect such misexpression such as genetic lesions or mutations gene [sic] itself or direct or indirect TAJ gene regulatory sequences, the misexpression of genes or gene products which may in turn regulate TAJ expression or TAJ function that may be involved in any specific ectodermal dysplasia and determine if, for example, any of the above indicate a predisposition to a specific ectodermal dysplasia or indicates that one is suffering from a specific ectodermal dysplasia and further devise a course of treatment based on the work performed outside the disclosure of the instant specification." Answer, p.8, lines 7-15.

modulation with a normalizing "+++" designation. The qualitative "+++" indicates that unequivocal allele-specific expression modulation is obtained. Changes in TAJ expression are readily assayed by those skilled in the art, as taught and exemplified in our Specification, e.g. Example VI. Similarly, we believe the Answer's statement that the claims include methods of treating any TAJ disorder is not properly confined to the invention recited in our claims. Note that the entirety of the claimed modulating method is exemplified in Example VI.

Though there is no evidence to the contrary, we have provided an expert Declaration under 37CFR1.132 documenting that the Specification readily enables one of ordinary skill in the art to practice this modulation method as claimed without undue experimentation. Hence, the *uncontroverted* evidence of record demonstrates compliance with 35USC112, first paragraph.

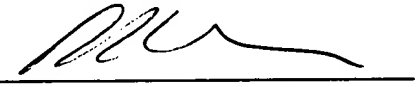
In his Answer, the Examiner again lashes at strawmen of his own construction. The claims do not require anyone to treat or cure any disease and the claims do not require any research into any, much less hundreds of diseases. As much as we appreciate the Examiner's zeal, he is not addressing the claims at issue nor are his repeated exaggerations and mistatements consistent with an effort to maintain an accurate record.

Finally, the Answer's Group of Claims does not accurately represent the record: our Appeal Brief does properly provide separate arguments (and in separate paragraphs) for each claim group.

Appellants respectfully request reversal of the pending Final Action by the Board of Appeals.

We hereby petition for and authorize charging to our Deposit Account No. 19-0750 all necessary extensions of time. The Commissioner is hereby authorized to charge any necessary fees (small entity) or credit any overpayments associated with this communication to our Deposit Account No. 19-0750 (order no. UTSD:0680).

Respectfully submitted,
SCIENCE & TECHNOLOGY LAW GROUP



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